

Thermophysiological Responses of Human Volunteers to Whole Body RF Exposure at 220 MHz

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Since 1994, our research has demonstrated how thermophysiological responses are mobilized in human volunteers exposed to three radio frequencies, 100, 450, and 2450 MHz. A significant gap in this frequency range is now filled by the present study, conducted at 220 MHz. Thermoregulatory responses of heat loss and heat production were measured in six adult volunteers (five males, one female, aged 24–63 years) during 45 min whole body dorsal exposures to 220 MHz radio frequency (RF) energy. Three power densities (PD = 9, 12, and 15 mW/cm² [1 mW/cm² = 10 W/m²], whole body average normalized specific absorption rate [SAR] = 0.045 [W/kg]/[mW/cm²] = 0.0045 [W/kg]/[W/m²]) were tested at each of three ambient temperatures (T_a = 24, 28, and 31 °C) plus T_a controls (no RF). Measured responses included esophageal (T_{esoph}) and seven skin temperatures (T_{sk}), metabolic rate (M), local sweat rate, and local skin blood flow (SkBF). Derived measures included heart rate (HR), respiration rate, and total evaporative water loss (EWL). Finite difference-time domain (FDTD) modeling of a seated 70 kg human exposed to 220 MHz predicted six localized ‘hot spots’ at which local temperatures were also measured. No changes in M occurred under any test condition, while T_{esoph} showed small changes (≤ 0.35 °C) but never exceeded 37.3 °C. As with similar exposures at 100 MHz, local T_{sk} changed little and modest increases in SkBF were recorded. At 220 MHz, vigorous sweating occurred at PD = 12 and 15 mW/cm², with sweating levels higher than those observed for equivalent PD at 100 MHz. Predicted ‘hot spots’ were confirmed by local temperature measurements. The FDTD model showed the local SAR in deep neural tissues that harbor temperature-sensitive neurons (e.g., brainstem, spinal cord) to be greater at 220 than at 100 MHz. Human exposure at both 220 and 100 MHz results in far less skin heating than occurs during exposure at 450 MHz. However, the exposed subjects thermoregulate efficiently because of increased heat loss responses, particularly sweating. It is clear that these responses are controlled by neural signals from thermosensors deep in the brainstem and spinal cord, rather than those in the skin. Bioelectromagnetics 26:448–461, 2005. Published 2005 Wiley-Liss, Inc.[†]

Key words: thermoregulation; body temperatures; sweating; thermal sensation; resonant frequency; deep thermosensors

INTRODUCTION

Since initiating a research program in 1994, we have learned much about how human beings respond in the presence of radiated radio frequency (RF) energy at levels that may heat the body tissues. However, there is a significant gap in the range of frequencies tested. In the current effort, we measured human thermoregulatory responses to RF exposures at a frequency in the critical range of transition from deep body heating to more superficial energy deposition, 220 MHz. This fills the gap between our previous studies conducted at 100 and 450 MHz [Adair et al., 1998, 2003]. Tissue heating is the only established mechanism of interaction of RF

fields with the human body. Thus, the purpose of this research program is to quantify the basic physiological thermoregulatory responses of human volunteers during periods of controlled RF exposure under specific environmental conditions and at several RFs.

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Seven studies have been conducted in our laboratories, each using the identical protocol and methodologies. In all studies, seated subjects were exposed dorsally for 45 min to RF energy at frequencies of 100, 450, and 2450 MHz, at each of three ambient temperatures ($T_a = 24, 28, \text{ and } 31^\circ\text{C}$). Several levels of energy absorption were tested at each frequency, including levels that exceed the applicable safety standard [IEEE C95.1, 1999]. While warm environments combined with the higher SARs usually evoked increases in SkBF and vigorous sweating responses, in no case was an increase in core temperature greater than 0.2°C observed. In fact, in some cases, the thermoregulatory responses were so efficient that a reduction in core temperature was recorded. These observations, most of which have been published in peer-reviewed journals [Adair et al., 1998, 1999a,b, 2001a,b, 2003; Allen et al., 2003], have demonstrated that human thermoregulatory responses are more than adequate to cope with RF exposures, even when they exceed the maximum permissible exposure (MPE) specified in the safety standard.

Extrapolation of animal data to humans still remains uncertain, so it is essential to build some critical bridges between the responses of humans and animals. One means of achieving this goal is to collect selective, pertinent data on human volunteers under highly controlled conditions. Studies performed in our laboratories are the only work involving human physiological responses to RF energy being conducted anywhere in the world. During passive exposure of a human being to a thermogenic RF field, the energy may be deposited in specific locations within the body's tissues. As is well known, the pattern of energy deposition will depend on many physical attributes of both the RF source and the biological target.

The research reported in this study involves one frequency (220 MHz) in addition to those already studied. At this frequency, close to the upper end of the human resonance range, RF energy may be deposited somewhat more superficially than at 100 MHz but deeper than at 450 MHz. As was the case for the 100 MHz study, subjects were exposed over their whole body while seated inside a unique anechoic chamber [Adair et al., 2003; Allen et al., 2003]. RF energy at 220 MHz also penetrates below the skin surface and can heat some deep tissues and organs directly. Using the same temporal protocol as in all of our previous work, we included some PD that provide the same rate of RF energy absorption as previously studied. We predicted that local sweating rate and SkBF would increase at higher rates in all test environments and that little or no increase in esophageal temperature would occur because the mobilized heat loss responses

would function efficiently to prevent a rise in deep body temperature.

METHODS

Subjects

The subjects were six adult volunteers, five males and one female (age range: 24–63 years; height range: 1.65–1.88 m; weight range: 61.5–100.3 kg). Details for individual subjects, who were drawn principally from Brooks City-Base personnel, are shown in Table 1. In the Table, A_D is the skin surface area, calculated from the equation provided in the table [DuBois and DuBois, 1916]. The last column of the table shows the seated height (sith) of each subject. All were in excellent health and most exercised regularly. Before testing began, each subject was fully briefed on the details of the experiment and the level of risk. Each signed an informed consent document that had been approved by the Wright-Patterson AFB Institutional Review Board (IRB) and the Office of the Air Force Surgeon General. Subjects were not paid for their participation and were blind to the details of individual tests. They were allowed to read or listen to music during each test session and were reminded frequently that they could terminate their participation at any time without penalty.

Test Chamber, RF Source, Field Measurements, and Dosimetry

The experiments were conducted in a $6.7 \times 6.7 \times 9.8$ m ($22 \times 22 \times 32$ ft) electrically shielded anechoic chamber; all interior walls were lined with 1.83 m (6 ft) pyramidal microwave absorber. The door to the chamber, located 2.13 m (7 ft) above the floor of the building, was accessed by an inclined ramp to a platform at door level. A horizontal Fiberglass™ grid deck inside the chamber allowed placement of antenna (tunable dipole in a vertical 90° corner reflector) and chair for the test subject. Stations for test equipment, computers, audio and video links to the subject, research personnel, etc., were located outside the chamber on the platform, in close proximity to the subject (cf. Fig. 1 in

TABLE 1. Characteristics of the Six Subject Volunteers

Subject	Sex	Age	h (m)	m (kg)	A_D (m ²)	sith (m)
SA	M	63	1.88	101.9	2.28	1.32
DB	M	61	1.85	103.3	2.26	1.31
SD	M	53	1.70	78.6	1.89	1.27
WH	M	60	1.83	83.6	2.05	1.33
AL	M	33	1.80	82.7	2.02	1.27
VS	F	24	1.65	61.5	1.67	1.23

h, standing height; m, mass; sith, sitting height.
 $A_D, 0.202 \text{ m}^{0.425} \text{ h}^{0.725}$.

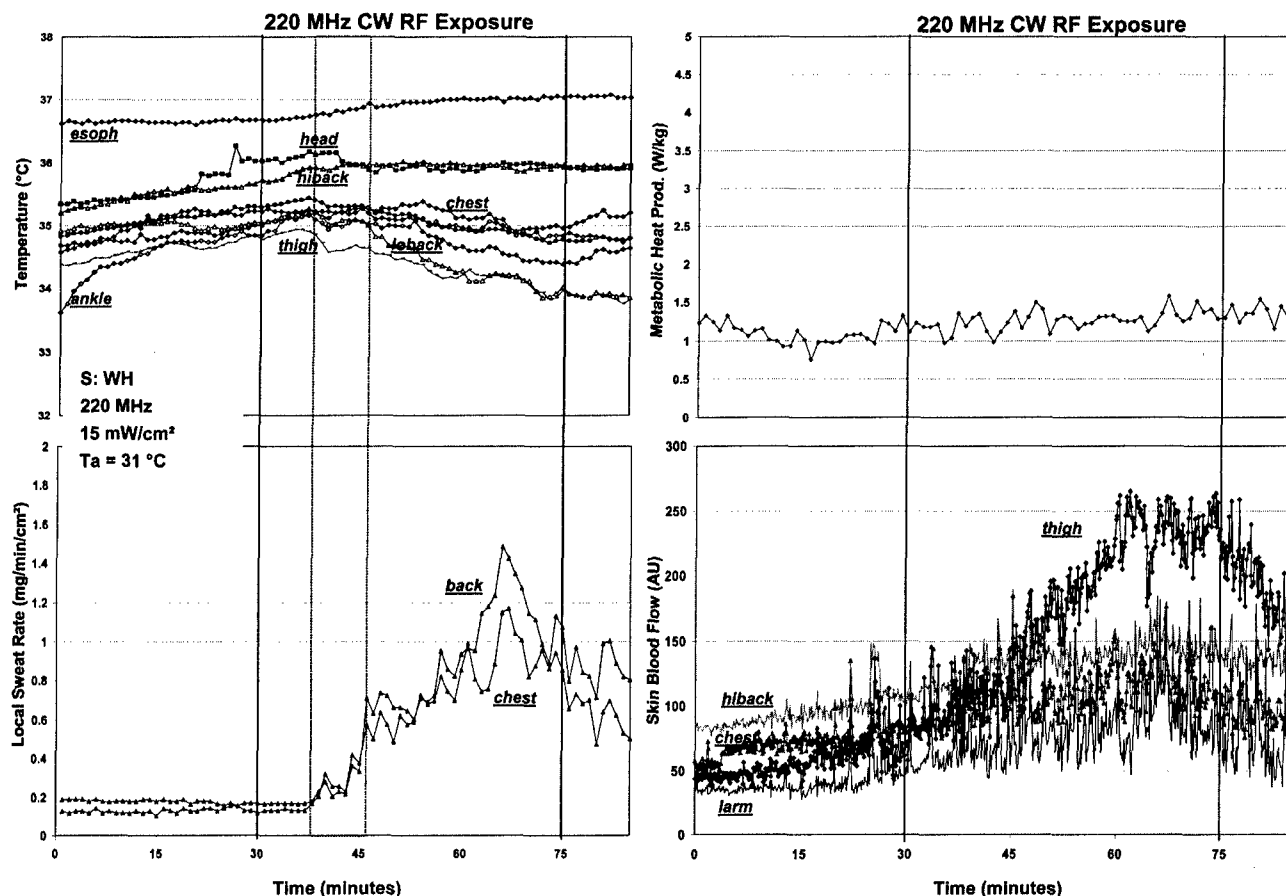


Fig. 1. Raw data collected on one male subject (WH) during a single test that involved a 45 min dorsal exposure of the whole body to 220 MHz continuous wave (CW) radio frequency (RF) energy at $PD = 15 \text{ mW/cm}^2$ in $T_a = 31^\circ\text{C}$. Heavy vertical lines at 30 and 75 min delineate period of RF exposure. Dashed vertical lines indicate relationship between core temperature (T_{esoph}) and sweating response (see text). Data are plotted as a function of time (min). **Upper left:** Esophageal temperature and seven skin temperatures ($^\circ\text{C}$); **lower left:** local sweat rate from chest and back (mg/min/cm^2); **upper right:** metabolic heat production (W/kg); **lower right:** local skin blood flow (SkBF) at four sites on back, thigh, forearm, and chest (arbitrary units, AU). [The color figure for this article is available online at www.interscience.wiley.com.]

Adair et al., 2003). The RF source was an Amplifier Research Model 2000LA transmitter located outside the chamber at the foot of the ramp. The maximum output power of this source was 2.3 kW at 220 MHz in the CW mode. Calculations based on the characteristics of this exposure system showed the far field to be at any distance greater than 1.5 m from the dipole.

The RF field encompassed the total volume of the seated subject. It was mapped across three $80 \times 80 \text{ cm}$ planes (located 2.0, 2.25, and 2.5 m from the dipole) with National Bureau of Standards (NBS, now NIST) E field and H field probes. The PD across the dorsal aspect of a seated standard human, as measured with the NBS E field probe at 2.25 m from the dipole antenna, is illustrated in Figure 5 of an accompanying study [Allen et al., 2005]. The average PD across the subject was

calculated as $11.6 \pm 0.53 \text{ mW/cm}^2$ ($116. \pm 5.3 \text{ W/m}^2$) for 1.0 kW forward power. The finite difference-time domain (FDTD) method was applied to a seated version of the 3-D Brooks AFB human dosimetry model [<http://www.brooks.af.mil/>] to calculate the normalized whole body SAR for a 70 kg man. The result was $0.70 \text{ (W/kg)/(mW/cm}^2)$ [$0.07 \text{ (W/kg)/(W/m}^2)$].

Dosimetric measurements on a 64.4 kg soft plastic man model [Olsen and Griner, 1989] filled with muscle equivalent material [Guy, 1971] were made with the model seated in the plastic chair at the subject's location inside the test chamber. Local SAR measurements were made with nonperturbing Vitek (BSD Corp., Salt Lake City, UT, USA) temperature probes inserted to several depths at eight locations (head, neck, chest, pelvis, arm, thigh, calf, and foot) of the model. Two complete sets of

measurements were made and then averaged. The total absorbed power, based on the sum of the eight contributing partial-body masses, was 50.5 W at a PD of 17.4 mW/cm². The average whole body SAR was determined to be 0.78 W/kg and the average normalized SAR was 0.045 (W/kg)/(mW/cm²) [Durney et al., 1986]. For details of the empirical and theoretical dosimetry, see Allen et al. [2005].

Physiological Response Measures

During the experimental tests, many heat production and heat loss responses were measured. Core and skin temperatures were monitored with a Fiso Technologies, Inc. UMI 8 instrument (Quebec City, PQ, Canada) fitted with fiberoptic temperature probes (Models FOT-m/2m or FOT-L-2m-PEEK). Core body temperature (T_{esoph}) was measured with a Fiso probe inserted into a nasally-introduced esophageal catheter to the level of the left atrium (catheter length to the external nares equal to one fourth the subject's height) [Wenger, 1983]. Temperatures at six skin sites were measured with Fiso probes that were held in place with perforated plastic tape. These sites were the anterior right thigh, left upper chest, left forearm, left upper back, central lower back, and central forehead. A mean skin temperature (\bar{T}_{SK}) was calculated from a weighted average of these six sites by the following equation:

$$\begin{aligned} \bar{T}_{\text{sk}} = & 0.18 T_{\text{arm}} + 0.23 T_{\text{leg}} + 0.21 T_{\text{forehead}} \\ & + 0.17 T_{\text{loback}} + 0.11 T_{\text{hiback}} + 0.10 T_{\text{chest}}. \end{aligned} \quad (1)$$

The individual weighting factors in Equation 1 are based on the product of regional area [Hardy, 1949] and local relative thermal sensitivity [Nadel et al., 1973]. Each temperature was sampled at 1 min intervals during the test. A Fiso temperature probe was also taped to the back of the left ankle, as was done in experiments at 100 MHz [Adair et al., 2003]; this measure was not included in Equation 1. A Vitek temperature probe was suspended beside the subject's chair to monitor local T_a continuously. Because FDTD modeling of a seated 70 kg human indicated several localized regions of high RF energy deposition at 220 MHz, several other temperatures were measured with nonperturbing Luxtron fiberoptic temperature probes (Mountain View, CA). These locations were the base of the skull, kidney region, back of the knee, front of the ankle, side of the neck, and top of the foot.

Metabolic heat production, M (W/kg), was calculated from continuous recordings of the fractions of O₂ and CO₂ in the expired air and the expired ventilatory volume. Local sweat rate (\dot{m}_{sw}) was measured from the left chest and left upper back with ventilated Plexiglas

capsules (area = 12.8 cm²) secured to the skin with medical adhesive. Chamber air was drawn through the capsules at ~1.7 L/min and thence outside the chamber. The increase in dewpoint temperature (T_{dp}) of capsule air, with respect to T_{dp} of chamber air, was the basis for calculating \dot{m}_{sw} [Adair et al., 1998]. Local SkBF was monitored continuously at three sites (left forearm, right thigh, and left upper back) by FLOLAB laser-Doppler flowmeters (Moor Instruments Ltd., Devon, UK). In 29 of the 84 total tests, a fourth flowmeter was available to measure SkBF on the right chest. HR, derived from pulsing SkBF records, was recorded eight times during each test.

Test Protocol

Before each test, the subject emptied the bladder, put on a bathing suit, inserted the esophageal catheter in accordance with previous instructions, and was weighed on a sensitive platform scale (accurate to 1 g). State of hydration was not regulated. Inside the anechoic chamber, the subject sat on a light plastic chair, elevated 53.8 cm (21.2 in) above the grid floor, facing the rear chamber wall. The Fiso and Luxtron temperature probes, laser-Doppler flowmeter probes, and sweat capsules were secured to the skin and a Fiso probe was inserted into the esophageal catheter and taped in place. A light plastic mask was placed over the subject's face and secured with Velcro straps. After the O₂/CO₂ system was calibrated, a flexible Teflon hose was connected to the mask to carry the expired air outside the chamber for analysis. The hose was supported by a cord attached to a plastic frame above the subject's head, thereby reducing the strain on the subject's neck. Several category scales were displayed on a Styrofoam panel directly in front of the subject so that he/she could make judgments of sensations and thermal comfort at intervals during the test (cf. Table 2 in Adair et al., 2001b). Two investigators were in constant visual and voice contact with the subject during the test session. Because the test protocol was judged to be of more than minimal risk, a medical monitor was either present or on call during every test.

Each test began with 30 min of equilibration to the prevailing T_a (24, 28, or 31 °C). The subject was then

TABLE 2. Group Mean Change in Heart Rate (HR) During RF or Sham Exposure as a Percentage of Preceding 30 min Baseline Period for all Test Conditions

T_a (°C)	Power density (mW/cm ²)			
	0	9	12	15
24	-1.5%	3.0%	6.9%	9.1%
28	1.7%	8.1%	9.2%	14.3%
31	7.1%	7.8%	7.2%	10.1%

exposed for 45 min to 220 MHz CW energy at a specific PD (9, 12, or 15 mW/cm²) or sham exposed (no RF). A 10 min re-equilibration completed the test, which lasted a total of 85 min. Category judgments of thermal sensation, thermal comfort, perceived sweating, and thermal acceptability were taken at 25, 45, 65, and 80 min of the test. Subjects were also encouraged to report any unusual sensations at any time. After the chamber was opened, the subject was debriefed while the mask and other instrumentation were removed. The subject was immediately weighed to determine total EWL, after which he/she gently pulled out the esophageal catheter. The entire procedure required about 2 hours.

Data Analysis

A total of 72 test sessions was conducted. The individual physiological responses of each subject

under each specific test condition were plotted in real time to look for irregularities in recordings, for example, missing data points, baseline shifts, etc., and to discover general trends in individual responses. These responses included six skin temperatures and weighted mean skin temperature (Eq. 1), esophageal temperature (T_{esoph}), ankle temperature, six temperatures at predicted hot spots, metabolic heat production (M), sweating rate (m_{sw}) from back and chest, and SkBF at three or four sites.

The data for all subjects were then pooled for each test condition and grand means for each response were calculated and plotted as a function of time. For these group data, mean changes (± 1 SD) in each measured response were calculated for each test condition as follows: the mean of the final 10 min of the initial 30 min equilibration period was subtracted from the mean of the final 10 min of the RF exposure period to yield the

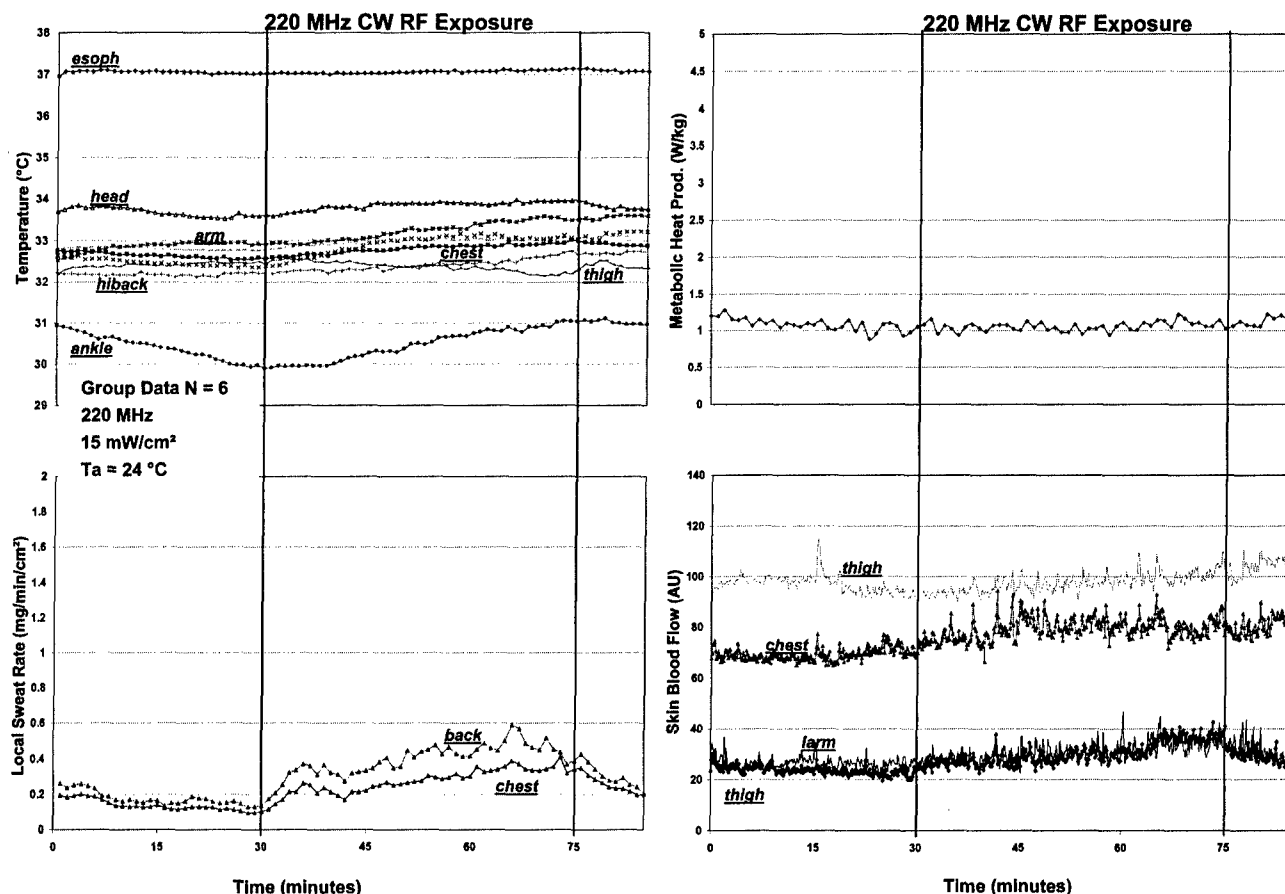


Fig. 2. Mean data for the group of six subjects exposed to 220 MHz CW RF energy at PD = 15 mW/cm² in $T_a = 24^\circ\text{C}$. **Upper left:** Esophageal temperature, six skin temperatures + ankle temperature ($^\circ\text{C}$); **lower left:** local sweat rate from chest and back (mg/min/cm²); **upper right panel:** metabolic heat production (W/kg); **lower right:** local skin blood flow at four sites, left forearm, right thigh, left upper back, and left upper chest (AU). [The color figure for this article is available online at www.interscience.wiley.com.]

mean steady-state change in response attributable to RF exposure. Control (no RF) data were similarly analyzed at comparable time periods, as were changes in HR. In similar fashion, mean judgments of thermal sensation, thermal comfort, thermal preference, and perception of sweating were calculated across subjects for the four inquiries during each test session. Total EWL was determined from pre- and post-test body weights, for comparison with measured rates of sweating on the back and chest in individual experiments. Additional statistical treatments, appropriate to selected comparisons, are described below in the Results section.

RESULTS

Physiological Responses

Figure 1 shows data from a single experimental test on one male subject (WH) at $T_a = 31^\circ\text{C}$, which incorporated a 45 min exposure to RF energy at a $PD = 15\text{ mW/cm}^2$, indicated by the two vertical lines on each panel. The two left panels show T_{esoph} , six T_{sk} , and

the weighted mean skin temperature (T_{sk}) (upper) and local m_{sw} on back and chest (lower). Also shown in the upper panel is the ankle skin temperature (T_{ankle}). The two right panels of the figure show M (upper) and local SkBF at four sites (lower). During the initial 30 min equilibration to this warm environment, individual T_{sk} slowly increased to relatively stable levels, T_{esoph} changed little, m_{sw} and M were at low levels, and SkBF rose slightly. The onset of the 220 MHz field initially produced a slow rise in body temperatures and SkBF for ~ 8 min, at which time m_{sw} began to increase. After another 7 min, T_{esoph} reached 36.9°C and m_{sw} increased sharply. Thereafter, all T_{sk} began to fall, due to evaporative cooling, SkBF increased at most sites, and T_{esoph} stabilized at $\sim 37^\circ\text{C}$, the nominal threshold for sweating in most adult humans. No significant change in M was evident during the test. Unlike data reported for 100 MHz [Adair et al., 2003], no change occurred in T_{ankle} during the 45 min RF exposure period.

Figures 2–4 show group mean data ($N = 6$) for tests involving a 45 min RF exposure at a $PD = 15\text{ mW/cm}^2$,

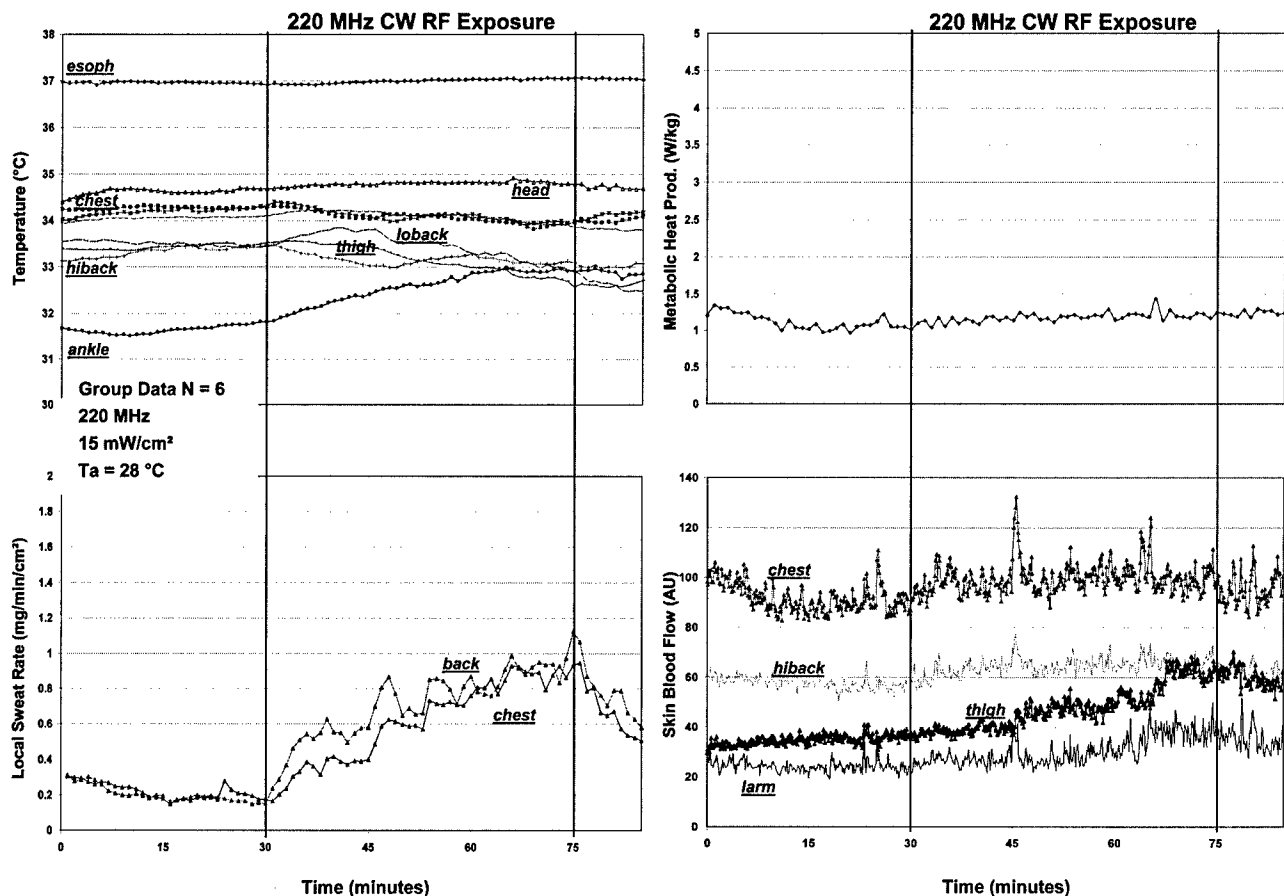


Fig. 3. Mean data for the group of six subjects exposed to 220 MHz CW RF energy at $PD = 15\text{ mW/cm}^2$ in $T_a = 28^\circ\text{C}$, plotted as a function of time (min). The four panels are the same as for Figure 3. [The color figure for this article is available online at www.interscience.wiley.com.]

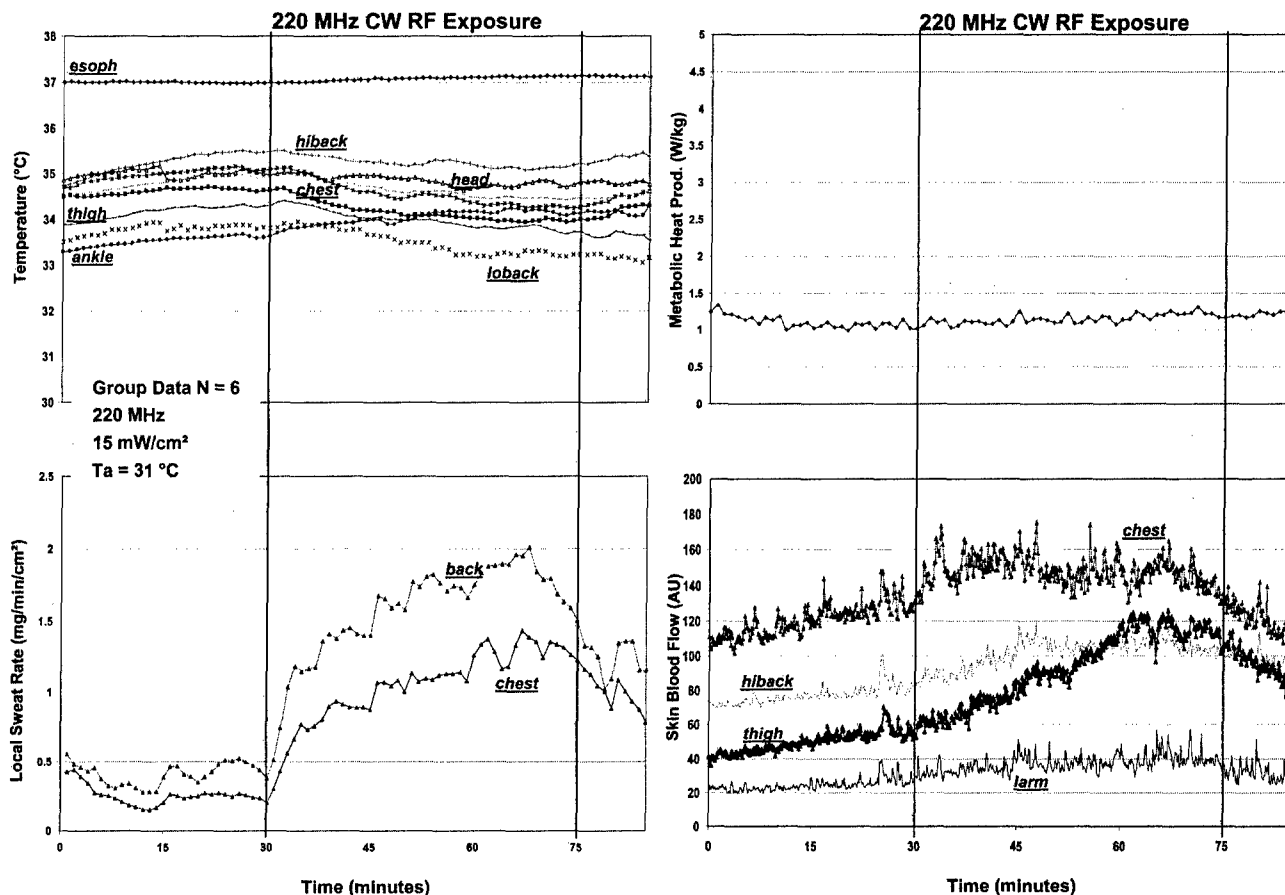


Fig. 4. Mean data for the group of six subjects exposed to 220 MHz CW RF energy at PD = 15 mW/cm² in $T_a = 31^\circ\text{C}$, plotted as a function of time (min). The four panels are the same as for Figures 3 and 4. [The color figure for this article is available online at www.interscience.wiley.com.]

presented at $T_a = 24, 28$, and 31°C , respectively. The format of each figure is similar to that of Figure 1; body temperatures and local \dot{m}_{sw} are shown in the left panels, while \dot{M} and local SkBF are shown in the right panels. Across all T_a , both \dot{M} (upper right panel) and T_{esoph} (upper left panel) were essentially constant at the level characteristic for each dependent variable. In fact, the mean ΔT_{esoph} across the 45-min RF exposure was 0.1°C at 24°C , 0.14°C at 28°C , and 0.15°C at 31°C . Various measures of T_{sk} , such as forehead, chest, and upper back, remained relatively stable during the RF exposure at $T_a = 24^\circ\text{C}$, but fell during RF exposure at $T_a = 28$ and 31°C because of evaporative cooling due to sweating. Local SkBF measured at four sites on the body tended to rise more in the warmer environments, but the increase during RF exposure at 15 mW/cm^2 was similar across all T_a . It is clear that increases in SkBF during RF exposure contributed little to body heat loss in these experiments.

The most dramatic changes were measured in the sweating response. While the change in local back \dot{m}_{sw}

at $T_a = 24^\circ\text{C}$ was only 0.25 mg/min/cm^2 across the 45 min RF exposure, the same measure was 0.97 and 1.26 mg/min/cm^2 at $T_a = 28$ and 31°C , respectively. It is of interest that at $T_a = 31^\circ\text{C}$, sweating rates on both back and chest decreased toward the end of the RF exposure period, a response change that effectively stabilized the skin and esophageal temperatures. It is also clear that an increase in \dot{m}_{sw} occurred during the first minute of the RF exposure at 15 mW/cm^2 and the magnitude of this increase is a direct function of the prevailing T_a . Thus, the $\Delta\dot{m}_{sw}$ in the first minute of RF exposure was 0.04 mg/min/cm^2 at $T_a = 24^\circ\text{C}$, 0.1 mg/min/cm^2 at $T_a = 28^\circ\text{C}$, and 0.17 mg/min/cm^2 at $T_a = 31^\circ\text{C}$. It is abundantly clear that the rapid increase in sweating at RF onset is not related to any other measured variable, even in the warm T_a .

Figure 5 shows the group mean $\Delta\dot{m}_{sw}$ for upper back (left panel) and chest (right panel) across the 45 min RF exposure for the 3 PD studied in the 3 T_a . Sham conditions (PD = 0) are also included in each panel. In general, sweating was somewhat greater on

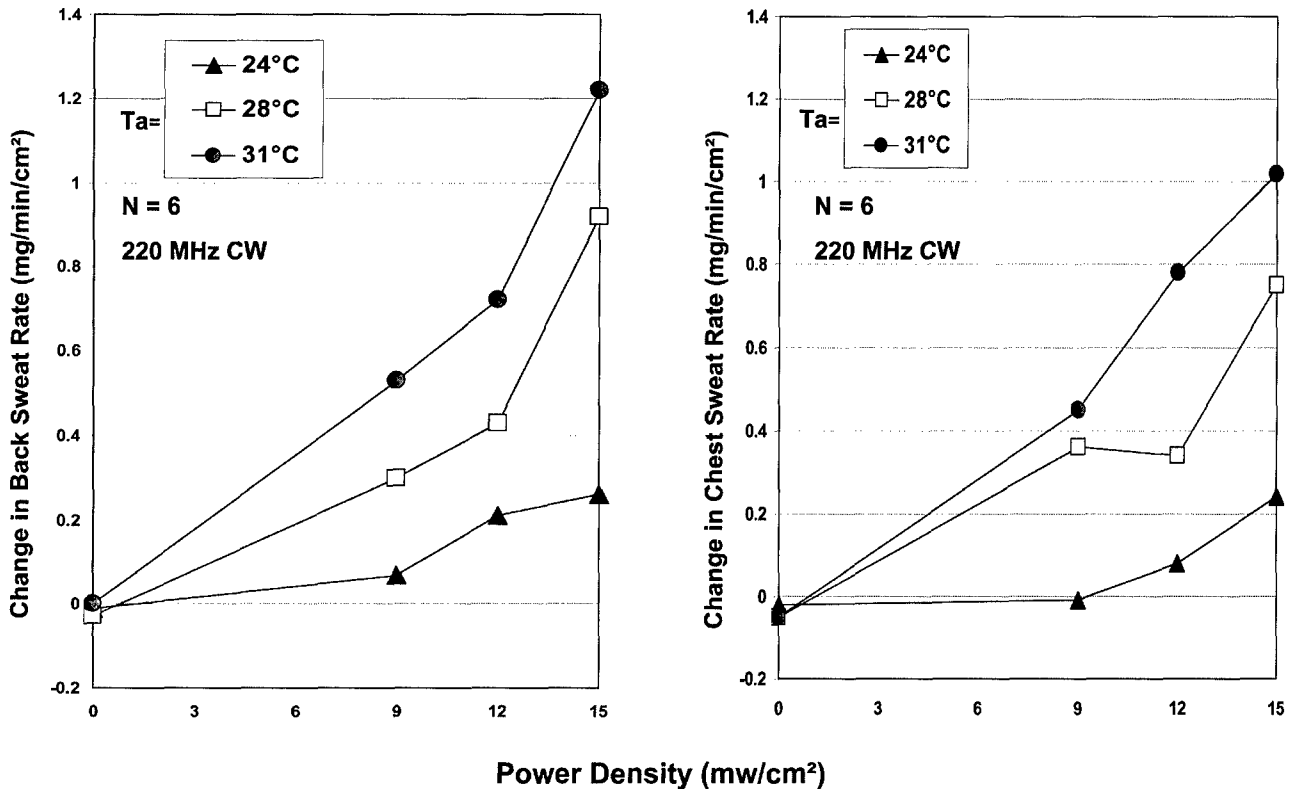


Fig. 5. Group mean changes in local sweat rate (mg/min/cm^2), measured from the end of the 30 min baseline to the end of the 45 min whole body exposures to RF (or sham) exposure, plotted as a function of PD (mW/cm^2). The parameter is ambient temperature. Back sweat rate (**left panel**) and chest sweat rate (**right panel**). Calculated SDs were smaller than the symbols used to plot the data. [The color figure for this article is available online at www.interscience.wiley.com.]

the back than on the chest but the functions in both panels are remarkably similar. Under sham conditions, $\Delta\dot{m}_{\text{sw}}$ on back and chest, measured between min 30 and 75 of the test, was essentially zero at all T_a . It appears that the major finding of this study conducted at 220 MHz is that the core body temperature, measured in the esophagus at the level of the heart, was efficiently regulated by heat loss through sweating under all conditions tested, even at the highest PD in the warmest environment.

HR was recorded at ~ 15 min intervals during each test session. The range of baseline HR across individual subjects was 52.6–79.3 bpm, with a group mean of 69.6 bpm across all 30 min baseline periods. This rate is very close to the 72 bpm value for normal adults at resting M [Guyton and Hall, 1996]. Small HR changes during RF (or sham) exposures occurred as a result of increases in T_a or increases in PD at any given T_a . Table 2 shows the group mean change in HR, attributable to RF (or sham) exposure, as a percentage of the preceding baseline value. At all T_a , the greatest percentage change in HR occurred at the highest PD. In general, the increases in HR measured in this study were substantially higher than comparable increases

measured in our previous study conducted at 100 MHz [Adair et al., 2003]. This result may reflect differences in individual subjects, only four of whom participated in both studies.

Total EWL was calculated from pre- and post-test body weights and then compared with local sweat rates for each test. In general, total EWL was 60–70 g for non-sweating subjects, representing the level of insensible perspiration over a ~ 2 h period. Respiratory EWL would contribute no more than 10% of the total. For subjects who were sweating heavily, especially at $\text{PD} = 12$ and 15 mW/cm^2 in $T_a = 31^\circ\text{C}$, total EWL ranged as high as 320 g. These values are very similar to those reported for whole body RF exposure at 100 MHz [Adair et al., 2003].

Sensory and Comfort Judgments

The judgments of thermal comfort, thermal sensation, perceived sweating, and thermal preference were taken four times (Trials 1–4) during each test, at min 25, 45, 65, and 80. The subjects responded in terms of six category scales [cf. Table 2 in Adair et al., 2001b]. As was the case in the study at 100 MHz [Adair et al., 2003], judgments of thermal sensation changed little

TABLE 3. Change in Category Judgments of Thermal Comfort, Thermal Sensation, Perceived Sweating, and Thermal Preference Resulting From Radio Frequency (RF) or Sham Exposure

	Δ Thermal comfort	Δ Thermal sensation	Δ Perceived sweating	Δ Thermal preference
Sham				
$T_a = 24^\circ\text{C}$	0.00 (0.00) ^a	0.00 (-0.33)	0.00 (0.00)	0.00 (0.17)
$T_a = 28^\circ\text{C}$	0.08 (0.17)	0.08 (0.25)	0.08 (0.17)	0.00 (-0.50)
$T_a = 31^\circ\text{C}$	0.00 (0.17)	0.00 (0.75)	0.25 (1.17)	0.00 (-0.33)
9 mW/cm ²				
$T_a = 24^\circ\text{C}$	0.00 (0.17)	0.92 (0.42)	0.25 (0.33)	-0.33 (-0.17)
$T_a = 28^\circ\text{C}$	0.25 (0.58)	0.75 (1.33)	1.50 (2.00)	-0.50 (-0.83)
$T_a = 31^\circ\text{C}$	0.42 (1.00)	0.75 (1.75)	0.67 (2.08)	0.17 (-0.67)
12 mW/cm ²				
$T_a = 24^\circ\text{C}$	0.17 (0.17)	0.67 (0.25)	0.67 (0.75)	-0.33 (-0.17)
$T_a = 28^\circ\text{C}$	0.50 (0.50)	1.17 (1.17)	1.33 (1.50)	0.00 (-0.33)
$T_a = 31^\circ\text{C}$	1.00 (1.08)	1.25 (1.75)	2.08 (2.83)	0.00 (-0.83)
15 mW/cm ²				
$T_a = 24^\circ\text{C}$	-0.33 (0.00)	1.50 (0.67)	0.83 (0.83)	-0.50 (0.00)
$T_a = 28^\circ\text{C}$	0.58 (0.83)	1.08 (1.42)	1.58 (2.08)	-0.50 (-0.83)
$T_a = 31^\circ\text{C}$	0.75 (0.75)	1.42 (1.92)	1.92 (2.50)	-1.67 (-0.83)

^aTabulated data are group mean judgments ($n = 6$) in each category. Trial 1 was taken at min 25 and Trial 3 was taken at min 65 of test session. First number of each pair is the mean change in judgment: Trial 3-Trial 1. Second number, in parentheses, is group mean judgment on Trial 3 (see Table 2 in Adair et al., 2001b for details).

because little change occurred in T_{esoph} and most T_{sk} . On the other hand, there was a growing deterioration in thermal comfort, particularly at the higher PD in the warm T_a where sweating increased substantially. A summary of the most important findings, calculated in terms of response change (Trial 3 minus Trial 1), appears in Table 3.

Temperatures at Predicted "Hot Spots"

FDTD modeling of a 70 kg seated human indicated that during RF exposure at 220 MHz, 6 locations on the body appeared to have local SARs greater than 0.8 W/kg, which could be classified as "hot spots" [Allen et al., 2005]. During all tests conducted on the six subjects, Luxtron fiberoptic temperature probes were taped in place at these locations (top of foot, base of skull, back of knee, front of ankle, skin over the kidney, and side of neck). Measurements at each location were taken at 1 min intervals during the tests. Group mean temperature changes (min 75-30) in four of these predicted "hot spots" (top of foot, base of skull, back of knee, and front of ankle) are shown in the four panels of Figure 6. Each panel presents data for 3 PD + sham exposure at each of the 3 T_a . In general, data for $T_a = 24$ and 28°C gave orderly increases in local temperature, while at $T_a = 31^\circ\text{C}$ the data tended to be compromised by the warm environment. This was especially true for the back of the knee and the front of the ankle, less so for the top of the foot and the base of the skull. Nevertheless, these local temperature

changes confirmed the FDTD predictions of "hot spots" at specific locations on the body.

DISCUSSION

The study reported here is the second in which human volunteers have undergone intentional whole body exposure to a RF frequency that is close to resonance. Based on our experience in the 100 MHz study [Adair et al., 2003], it was again important that the field be mapped with great care, the dosimetry be as extensive as possible, and all potential electrical artifacts be identified and eliminated. During the experimental tests of individual subjects, all equipment was calibrated daily and the output power of the transmitter was monitored continuously. The same physiological responses were measured as for the 100 MHz study, with the addition of special local temperature measurements to confirm predictions of localized "hot spots" as described above.

The results of the 220 MHz study were both similar and different from comparable results reported at 100 MHz [Adair et al., 2003]. During the 220 MHz exposures of individual subjects, T_{esoph} showed small changes ($\approx 0.35^\circ\text{C}$) and never exceeded 37.3°C . No changes in M occurred under any test condition. As with similar exposures at 100 MHz, local T_{sk} changed little. Modest increases in SkBF were often recorded, especially at the higher PD and in the warm T_a . At 220 MHz, vigorous sweating was mobilized rapidly at

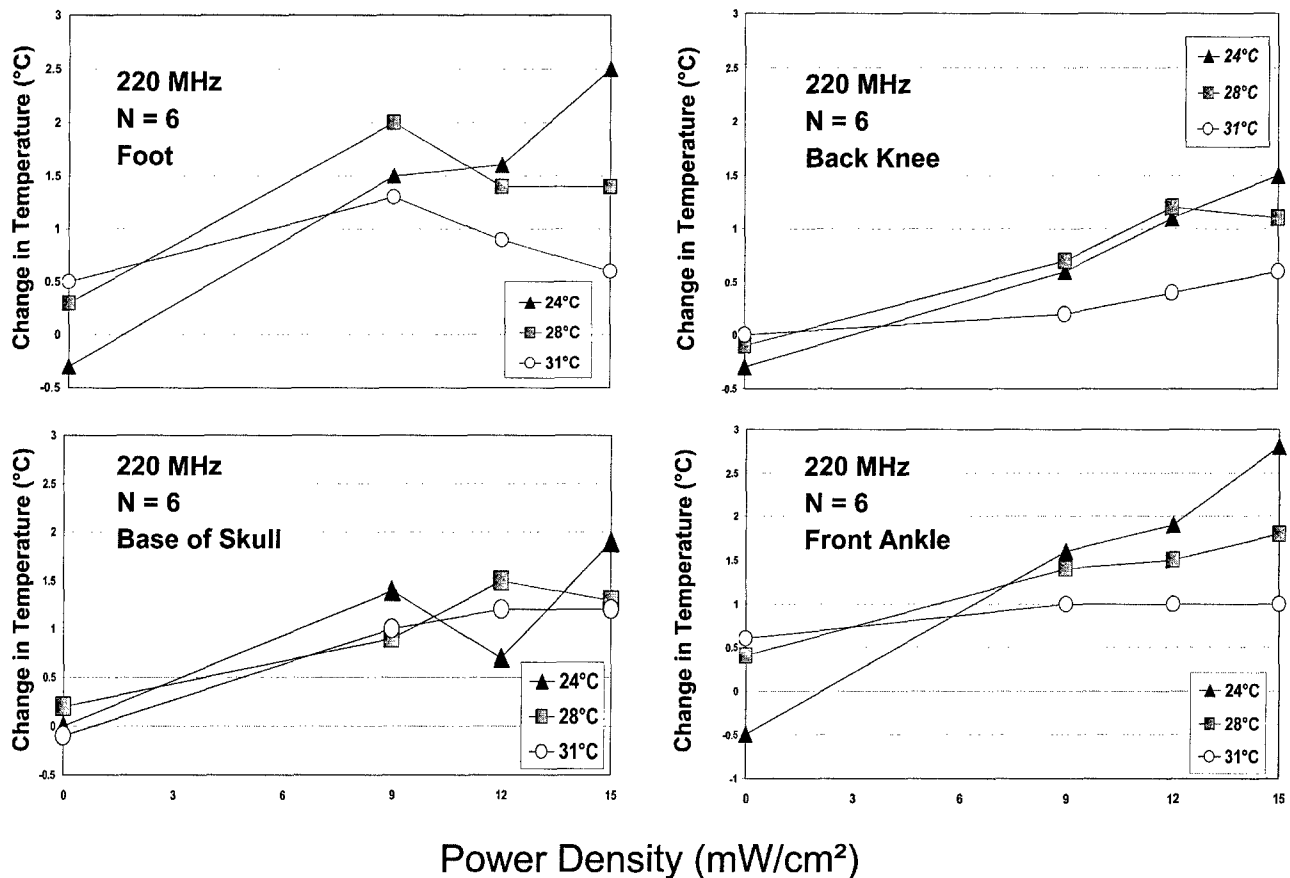


Fig. 6. Group mean changes in measured local "hotspots" (ΔT) that had been predicted by a FDTD model of a standard 70 kg seated man, during dorsal exposures to 220 MHz RF energy for 45 min. Local ΔT for the upper foot skin (upper left panel), base of skull (lower left panel), back of knee (upper right panel), and front of ankle (lower right panel) are plotted against power density (mW/cm^2) + sham (no RF). The parameter is ambient temperature (T_a). Calculated SDs were smaller than the symbols used to plot the data. [The color figure for this article is available online at www.interscience.wiley.com.]

PD = 12 and 15 mW/cm^2 and to higher levels than observed for equivalent PD at 100 MHz. Predicted "hot spots" were confirmed by local temperature measurements, although the temperature rise at the back of the ankle, so strong at 100 MHz, was less so at 220 MHz. The exposed subjects were found to thermoregulate most efficiently because of increased body heat loss, particularly through sweating.

The thermophysiological responses to whole body exposure at 220 MHz are similar in many respects to those reported for 100 MHz exposure [Adair et al., 2003]. For example, PDs of 6 and 8 mW/cm^2 at 100 MHz are equivalent to PDs of 9 and 12 mW/cm^2 at 220 MHz in terms of whole body SAR (Table 4).

Had we elected to study 10 mW/cm^2 at 100 MHz, the whole body SAR would have been identical with that of 15 mW/cm^2 at 220 MHz, namely 0.68 W/kg. These equivalences provide a basis for direct comparison of data between the two frequencies studied.

Careful examination of the group data for two equivalent PD at each frequency yielded some interesting conclusions. Esophageal temperature remained close to $37 \pm 0.15^{\circ}\text{C}$ throughout all tests at both frequencies, although the actual level varied slightly with individual subjects and the prevailing T_a . Those local skin temperatures that contributed to the weighted average \bar{T}_{sk} (Eq. 1) were T_a dependent at both frequencies, but evidence for evaporative cooling was

TABLE 4. Power Density and SAR Equivalents at 100 and 220 MHz

	Power density (PD) (mW/cm^2)	Whole body SAR (W/kg)
100 MHz	6	0.41
	8	0.54
220 MHz	9	0.405
	12	0.54

stronger at 220 MHz, especially in the warmer T_a . The increase in ankle temperature was at least twice as great at 100 MHz than at 220 MHz, reflecting the predictions of FDTD modeling at the two frequencies. M was uniformly stable at ~ 1.2 W/kg at both frequencies and all PD tested. SkBF was quite variable, especially during RF exposure at 220 MHz, so that it was not possible to detect any specific trends related to each frequency. On the other hand, the threshold for sweating and the magnitude of m_{sw} during RF exposure differed considerably at equivalent whole body SARs at the two frequencies, with 220 MHz generating greater sweating and earlier initiation of the sweat response than did 100 MHz under all T_a tested. Specific details are summarized in Table 5.

In the 100 MHz study, subjects were exposed at a lower whole body SAR (0.27 W/kg, 4 mW/cm²) than in the present study. At this low level, it was appropriate to look for thresholds of individual thermoregulatory responses, of which sweating was a primary candidate. Analysis of the group data showed no sweating during RF exposure at $T_a = 24^\circ\text{C}$, initiation of erratic sweating (at min 45 of the test) at $T_a = 28^\circ\text{C}$, and a clear sweat threshold (at min 33 of the test) at $T_a = 31^\circ\text{C}$ with pulsatile increases in m_{sw} up to 0.58 mg/min/cm² at min 75. Skin and esophageal temperatures remained steady during RF exposure at $T_a = 24$ and 28°C , while at $T_a = 31^\circ\text{C}$, T_{esoph} rose 0.1°C and T_{loback} fell 0.3°C due to evaporative cooling. On the other hand, T_{ankle} rose about 1.5°C in all T_a during RF exposure at 4 mW/cm², just as the FDTD modeling of a seated human had predicted [Adair et al., 2003].

The range of radio frequencies that are currently considered to cover human resonance extends nominally from 30 to 300 MHz [ANSI C95.1, 1982]. Current RF exposure standards [IEEE Std C95.1, 1999; International Commission on Non-Ionizing Radiation Protection (ICNIRP), 1998] recommend a reduced level of field strength across this range, for example, 1 mW/cm², for persons cognizant of their exposure to RF energy in a controlled environment. The present study may be regarded as an extension of the earlier published study at 100 MHz [Adair et al., 2003]. While 100 MHz is very close to resonance for seated adult humans, 220 MHz is close to the upper boundary of the resonance range. RF energy at both frequencies is absorbed in deep tissues of the body rather than on the body surface. This condition results in an absence of the thermal sensation that occurs at frequencies at and above 2 GHz, which would be derived from stimulation of temperature-sensitive neurons in the skin. Nevertheless, thermoregulatory responses are generated just as efficiently during exposure at resonance as at higher frequencies, because of the presence of similar temperature-sensitive neurons located variously in the brainstem, spinal cord, and elsewhere in the central nervous system (CNS) [Adair, 2000].

The rapid mobilization of sweating observed during the first minute of a 220 MHz RF exposure at 15 mW/cm² in all tests T_a (cf. Figs. 2–4) can only be understood in terms of the thermal stimulation of temperature sensitive neurons residing in critical regions of the brainstem and spinal cord. Calculations of localized RF energy deposition in the body, through

TABLE 5. Thresholds for Sweating and Sweating Characteristics for Equivalent Whole Body SARs at 100 and 220 MHz and Three Ambient Temperatures, Based on Group Means

100 MHz			220 MHz		
	Threshold	Sweating		Threshold	Sweating
[†] 6 mW/cm ²			[†] 9 mW/cm ²		
$T_a = 24^\circ\text{C}$	None	Steady at 0.07 mg/min/cm ²	$T_a = 24^\circ\text{C}$	min 42	Small rise to 0.2 mg/min/cm ²
$T_a = 28^\circ\text{C}$	min 37	Peak of 0.44 mg/min/cm ² at 45 and then fell	$T_a = 28^\circ\text{C}$	min 31	Steady rise to 0.57 mg/min/cm ² at min 75
$T_a = 31^\circ\text{C}$	min 31	Variable rise to 0.72 mg/min/cm ² at min 75	$T_a = 31^\circ\text{C}$	min 31	Steady rise to 1.25 mg/min/cm ² at min 75
8 mW/cm ²			12 mW/cm ²		
$T_a = 24^\circ\text{C}$	min 39	Steady rise to peak of 0.3 mg/min/cm ² at min 75	$T_a = 24^\circ\text{C}$	min 33	Steady rise to 0.34 mg/min/cm ² at 75 min after peak of 0.41 mg/min/cm ²
$T_a = 28^\circ\text{C}$	min 44	Noisy rise to peak of 0.55 mg/min/cm ² at min 75	$T_a = 28^\circ\text{C}$	min 31	Steady rise to peak of 0.8 mg/min/cm ² at min 75
$T_a = 31^\circ\text{C}$	min 36	Steady rise to peak of 0.7 mg/min/cm ² at min 75	$T_a = 31^\circ\text{C}$	min 31	Steady rise to peak of 1.3 mg/min/cm ² at min 70 then fall to 1.2 mg/min/cm ² at min 75

Note: RF exposure occurred from 30 to 75 min of the test protocol. The threshold for sweating was determined by the minute (min) at which RF stimulated sweating began. T_a = ambient temperature ($^\circ\text{C}$).

[†]See Table 4 for SAR (W/kg) equivalents of power densities.

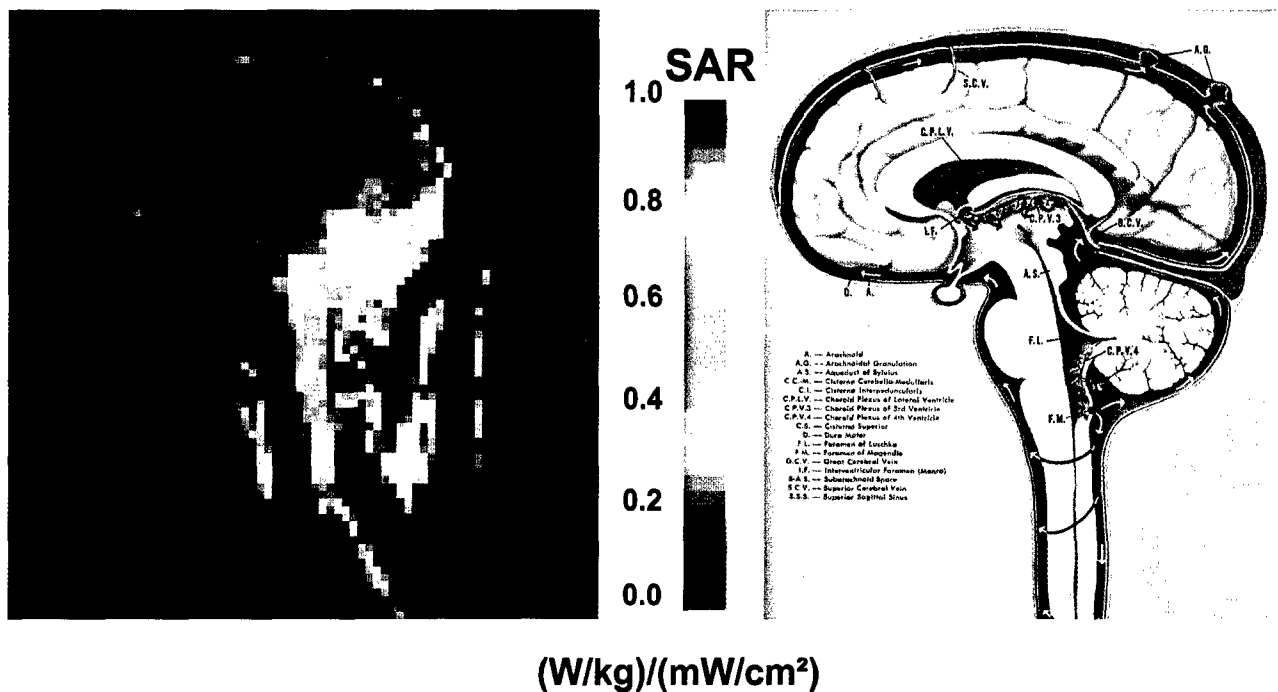


Fig. 7. Mid-sagittal sections of the human brain and spinal cord. **Left panel:** Shows FDTD model of 70 kg seated man exposed dorsally at 220 MHz; **right panel:** shows comparable anatomical structures [Netter, 1962]. The model indicates the normalized local SAR >1.0 [W/kg]/[mW/cm²] in cerebrospinal fluid that bathes the medial preoptic/anterior hypothalamic (PO/AH) and other brainstem nuclei that control thermoregulation during RF exposure at 220 MHz.

FDTD modeling, indicate that there is especially high power absorption (SAR >1.0 [W/kg]/[mW/cm²]) in the third and fourth ventricles, the medial preoptic/anterior hypothalamic nuclei (PO/AH), and the cerebrospinal fluid bathing these regions (Fig. 7). This is not surprising since the local power deposition, $P = E^2 \sigma$, is proportional to the conductivity of the local medium, which is a saline fluid with a high conductivity ($\sigma \sim 2.0$ at 100 MHz). Also, at 15 mW/cm², the local SAR is at least 15 W/kg and the temperature rise converts to 0.26 °C/min.

Our collective data [Adair et al., 2003; present study] indicate that since the core temperature of the body is held to within ± 0.2 °C during the 45 min period during which RF power is absorbed, the central temperature receptors must be sensitive to changes of less than 0.2 °C. In addition, these sensors respond in less than 1 min [Hensel and Kenshalo, 1969; Hensel, 1981]. On the other hand, since the diurnal cycle changes the core body temperature by about 1.0 °C over the day without the intercession of special heat loss systems to cool the body, the central temperature receptor systems must not respond to very slow temperature changes [Adair et al., 2003].

We assume that the central temperature sensors are similar to those that reside in the skin and generate sensations of warmth and cold. Thresholds of cutaneous

warmth sensation were determined for 16 adult males exposed to 10 s pulses of RF energy at five frequencies from 2.45 to 94 GHz [Blick et al., 1997]. Riu et al. [1996] used these data to solve the one dimensional bioheat equation [Gao et al., 1995] and thereby calculated the increase in skin temperature at incident powers generating the warmth thresholds. Their analysis showed that the thresholds are reached at a localized increase in temperature of ~ 0.07 °C at or near the skin surface. Others [Stolwijk and Hardy, 1966; Way et al., 1981; Stolwijk, 1983] have applied a physiological model of human thermoregulation to predict responses to environmental thermal stimuli. This model has recently been applied with considerable success to data derived from adult humans exposed to RF energy [Foster and Adair, 2004].

Starting in the mid 1960s, the use of implanted thermodes in the brain and spinal cord of assorted mammals allowed the recording of neural activity in CNS structures, for example, PO/AH, posterior hypothalamus, midbrain, spinal cord, medulla, etc. [Nakayama et al., 1963; Hardy, 1972; Hensel, 1981; Jessen, 1990; Boulant, 1996]. Recordings of firing rate versus local tissue temperature showed great sensitivity of individual warm-sensitive neurons in many of these structures. Like receptors in the skin, the central warm receptors appear to be differentiated C-fibers, unmye-

linated afferent nerve fibers of about 1–2 μm in diameter [Konietzny and Hensel, 1975, 1977]. At a normal body temperature ($\sim 38^\circ\text{C}$ for the cat), the mean firing rate (v) of axons from the warm receptors in the infraorbital area is about 7 pulses per second (pps), having a variation with temperature $dv/dT \sim 0.25$ pps/ $^\circ\text{C}$. Different fibers have firing rates that may differ by a factor of two, although the variation of the temperature dependence is somewhat less [Hensel and Kenshalo, 1969; Hensel, 1981].

Adair [2001] describes a physiologically plausible “voter coincidence” neural network such that secondary “coincidence” neurons fire upon the simultaneous receipt of sufficiently large sets of input pulses from pairs of primary neurons. The network operates so that the firing rate of the secondary, output neuron increases (or decreases) sharply when the mean firing rate of the primary neurons increases (or decreases) to a much smaller degree. In this way, signals, such as those from sensory systems that are manifest in very small increases in firing rates of sets of afferent neurons, can generate large changes in the firing rate of secondary “coincidence” neurons. These differential amplification systems can be cascaded to generate sharp “yes–no” signals that can direct physiological responses. Based on his analysis, Adair [2001] estimates that the information from 100 axons could sense a temperature change (ΔT) of about 0.03°C if the information were used efficiently. Such a small ΔT can easily be responsible for triggering central warm sensitive neurons to initiate appropriate heat loss responses, such as sweating, at the initiation of whole body RF exposure to 220 MHz, and in a very brief time, that is, less than 60 s.

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14. ABSTRACT Since 1994, our research has demonstrated how thermophysiological responses are mobilized in human volunteers exposed to three radio frequencies, 100, 450, and 2450 MHz. A significant gap in this frequency range is now filled by the present study, conducted at 220 MHz. Thermoregulatory responses of heat loss and heat production were measured in six adult volunteers (five males, one female, aged 24-63 years) during 45 min whole body dorsal exposures to 220 MHz radio frequency (RF) energy. The exposed subjects thermoregulate efficiently because of increased heat loss responses, particularly sweating. It is clear that these responses are controlled by neural signals from thermosensors deep in the brainstem and spinal cord, rather than those in the skin.					
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